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Thermal modelling of individual characteristics

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Als deze voorspellende waarde werd vergeleken met die van een multipele regressiemodel dat direct van de gebruikte data sets was afgeleid (= de "maximaal haalbare" voorspellende kracht), met individuele karakteristieken als onafhankelijke parameters en lichaamstemperatuur als afhankelijke, bleek dat het onafhankelijke computer model tussen 54 en 89% van de voorspellende waarde van het regressiemodel bezat. Behalve voor het koude klimaat, daar was deze verhouding gelijk aan nul. Als demonstratie van de mogelijkheden en de reacties van het model zijn enkele voorbeeldsimulaties toegevoegd.

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15. ABSTRACT (MAXIMUM 200 WORDS (1044 BYTES))

One of the major gaps in the prediction of heat stress response is the limited implementation of individual characteristics in prediction models. Without this individualization, the evaluation of the resultant average group response prediction necessitates the use of very conservative limit values for body temperature increase. This is caused by the wide range of responses observed within a group.

The present study aimed at the implementation of individual characteristics in a heat stress prediction model (THDYN), in order to investigate whether this would indeed result in a more precise prediction with less variance between predicted and observed responses. For this purpose, the relevant parameters related to anthropometric characteristics (body surface area [A_D], body tissue conductance, body heat capacity), sweating and skin blood flow control (training and acclimation) were introduced in the model. The parameters were derived from literature.

Next, data sets which were not used for the parameter estimation were used for a validation of the model changes. It was found that the individualized model indeed provided an improved prediction. The size of the improvement varied with the climate and the work type however. The best predictions for body heat storage were observed for fixed work loads in a warm humid and in a hot dry climate and for work loads relative to the individual maximum in a warm humid climate (Explained variance 27-53%). For relative work loads in a cool and in a hot dry climate the models predictive capacity for individuals was not significantly improved (<10%).

When this predictive power was compared to that of a multiple regression model derived from the data sets (the "maximal achievable" predictive power), using individual characteristics as independent parameters and body temperature as dependant variable, showed that the independent computer model had between 54 and 89% of the predictive power of the empirical model, except for the cool climate where this was zero.

It can be concluded that the additions to the model provide a good tool for study of effects of individual characteristics, but also that still a substantial part of individual variation is still not understood.

In order to demonstrate the model's possibilities and reactions, several example simulations are presented.

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Modellering van de individuele thermische reactie

G. Havenith

SAMENVATTING

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Als demonstratie van de mogelijkheden en de reacties van het model zijn enkele voorbeeld-simulaties toegevoegd.

1 INTRODUCTION

Numerical models of human responses to heat and cold exposure are widely used. Some of these models are empirical (Givoni & Goldman, 1972, 1973), others are restricted to heat balance calculations (ISO 7933, ISO TR 11079), or include a thermoregulatory system, including sweating and blood flow regulation (Nishi & Gagge, 1977; Stolwijk & Hardy 1977, Wissler, 1964, 1982) and some have added the physics of clothing (Werner 1989; Lotens, 1993; Werner & Webb, 1993). The validity of the predictions by various models is dependent on the combination of the climate, clothing and work load. One problem present in all models, however, is that even if their prediction is quite good for an average group response, the response of an individual may still show a large deviation from the prediction. The importance of having an insight in the mechanisms behind the individual differences will be discussed in the next paragraphs. The intention of the present paper is to incorporate individual aspects of thermoregulatory response in a prediction model, and to determine whether this actually improves the prediction of heat stress responses of specific individuals in a population.

1.1 Safety limits

Safety guidelines for occupational exposure to climatic stress have set the deep body temperature limit at 38°C (ISO 7933). This is by itself not a dangerous temperature at all. Many people reach this temperature riding their bicycle to work. Unfortunately, in the past many people have used this temperature as a limit value for each individual worker. This is not the correct way to use this limit, however. To understand this we have to go back to the origins of this value.

The most cited document for the 38°C limit is a document of the WHO: in 1969, the World Health Organisation (WHO, 1969) stated that it was inadvisable to allow deep body temperatures to exceed 38°C in prolonged daily exposure to heavy work. However, this document also states that under constant surveillance of the individual it is advised to stop exposures at 39°C. Further, both statements were based conditions where core temperature (measured by rectal temperature (T_{re})) supposedly is a direct function of metabolic rate and independent of ambient temperature (T_a) i.e. for exercise without severe external heat stress. Thus they may not necessarily be valid for heat exposures, where the increase in T_{re} is not only caused by metabolic heat production, but a hot climate too.

Data from Wyndham and coworkers (Wyndham et al., 1965) made them conclude that during heat exposures, values for T_{re} above 39.2°C should be considered excessive and may rapidly lead to total disability in most men due to excessive, often disturbing, physiological changes. From these sources, and the literature on heat stroke, two maximum T_{re}'s for an individual were adopted (Malchaire, 1996):

- 42°C as the maximum internal temperature to avoid physiological sequela (heat stroke),
- 39.2°C as the maximum temperature in normal work, to avoid heat exhaustion.

Doing equal work in equal climatic circumstances will not result in an identical T_{re} for all subjects, but in a Gaussian distribution of T_{re} (Havenith et al., 1997), which becomes positively skewed for longer heat exposures with high maximal T_{re}'s (Wyndham et al., 1965). In order to avoid some subjects to reach 39.2°C T_{re}, the average population T_{re} will therefore have to be much lower. Based on Wyndham's data, Malchaire (1996) analysed the chances for subjects to reach 39.2°C, for different mean T_{re} values for a group. He found that the chance for an individual to reach a T_{re} of 39.2°C was 10^{-4}, if the group mean was limited around 38°C, i.e. the WHO limit. Unfortunately, little information on the group characteristics for which the data were collected are available, so that these data are difficult to generalize to the full working population. Data from Havenith et al. (1990, 1995) where subject characteristics are well defined, will be analysed for the same purpose in the near future.

The approach followed by Malchaire gives a good idea of how the WHO may have wanted the limit of 38°C to be used: As long as the group mean does not exceed this value, it is unlikely that subjects at the extreme of the population will be at risk.

1.2   Safety limits in models

Predictive models, as e.g. ISO 7933 also aim at the prediction of a safe group response (i.e. 38°C maximal T_{re}) for 95 to 99% of the population. In the actual use of this model a different problem showed. When actual work places were evaluated, it was shown that according to ISO 7933 conditions in many workplaces in the mining industry were well above the models safety limits (Kampmann, 1992). However, at these workplaces few problems were encountered. One of the possible explanations is that workers at these locations are fitter than average and also acclimatized. Thus, the limit values for climatic stress used may be far too conservative for this special group. This situation is schematically presented in Figure 1. If the limit for the 95th percentile was set at 39°C, the distribution of observed core temperatures for an average population might look as presented. The fit, acclimatised miners would be situated at the left part of the distribution. It is clear that these are hardly stressed at all. The climatic stress limit for this group should be adjusted for the specific characteristics of this sub-population. The core temperature values of 39.2°C as limit and 38°C as mean may still be valid for the sub-group assuming a similar standard deviation in T_{re}'s, but conditions under which they will be reached are more severe than for the average population.
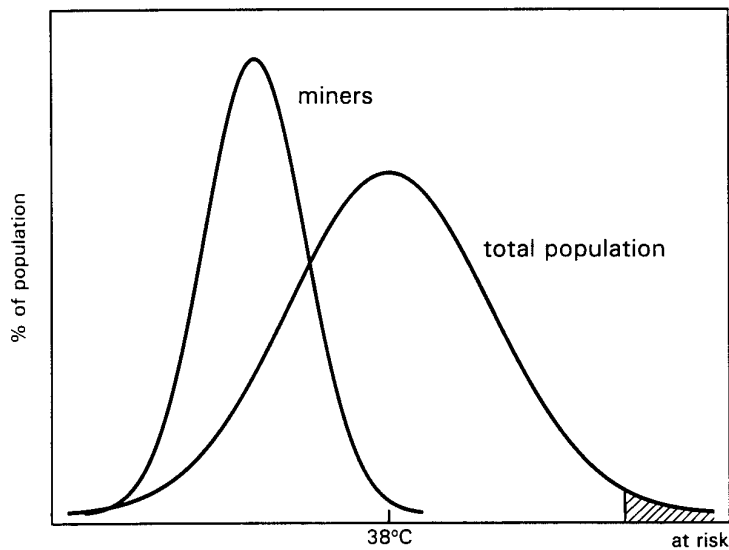
Fig. 1 Distribution of body core temperature in a general population exposed to identical external exercise and heat load. The subgroup data represents the results for the miners.

The problem present here is the struggle between safety for all on one hand and productivity on the other. Its basis is the lack of knowledge on how to adjust the safety limits to the individual.

From the above it is obvious that there is a need to produce better predictions of heat stress response at the level of an individual. Only then can limits be used with a proper balance of safety and productivity.

The purpose of the present paper is to incorporate individual characteristics in a thermoregulatory model (THDYN; Lotens, 1993) using data from literature, and to validate the individualized model with data from heat exposure experiments from our laboratory (Havenith & Van Middendorp, 1990; Havenith et al., 1995a,b; Havenith et al., 1997). The main research question is: "Can the prediction of heat strain by simulation models be improved by incorporation of individual characteristics".

2   THE MODEL

The model used as a starting point was described by Lotens (1993). It comprises a physiological part based on the Gagge (Gagge et al., 1971, 1986) two-node[1] model, and a physical model describing the heat transfer characteristics of clothing. The physiological model contains a number of control equations of physiological processes, as well as the heat transfer properties of the human body. The principle is represented in Figure 2.
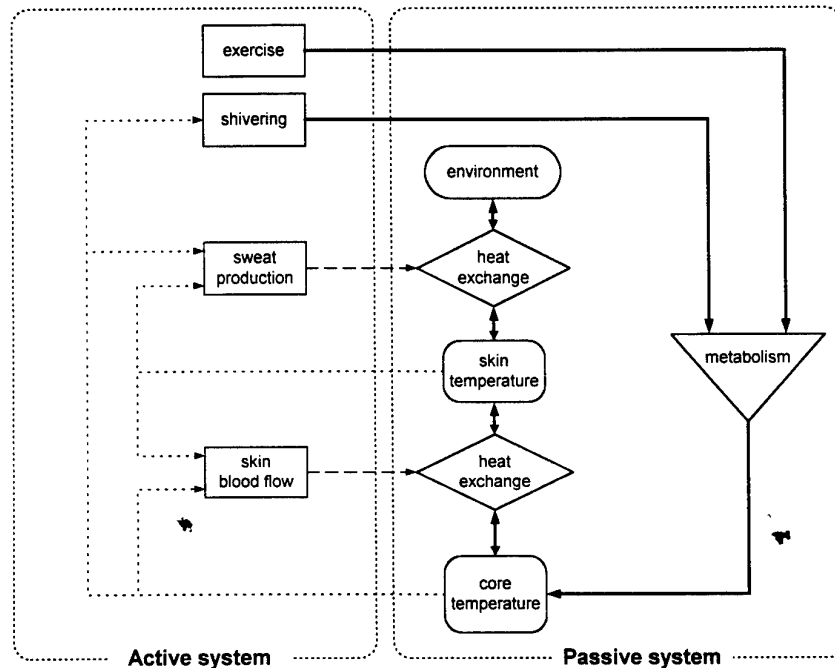

Fig. 2   Schematic representation of the physiological control system in the used computer model, the heat production inputs and the heat exchanges between body core and environment.

Core and skin temperature are used as input for several control systems (skin vasoconstriction/dilation, sweat production, shivering). The effector responses together with metabolism due to exercise result in a certain heat loss or gain, which then affects the passive system (the body), resulting in a new body temperature. The relation between effectors and resulting body temperature (the passive system) is affected by environmental parameters (heat transfer properties) and heat production levels (exercise). The passive system itself is defined in terms of heat capacity, mass and surface area which are constant in the Gagge model.

Individual differences may affect the control system as well as the passive system, as will be discussed in more detail later.

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[1] Two-node model: the nodes represent the body core and the body shell. The core being the compartment with the regulated and defended temperature, the shell being the buffer between core and environment, which temperature is determined by the heat exchanges with the core and with the environment.

3   INDIVIDUALIZING THE MODEL

Starting with the inventory of inter-individual differences in heat stress response by Havenith (1985), and surveying recent literature on these subjects, several additions and changes to the governing equations in the model will be made to "individualize" its response. The relevant model characteristics that will be discussed in this respect are: anthropometrics and adiposity, gender, age, sweat control and blood flow control.

3.1   Anthropometric characteristics and adiposity

The model currently mimics a standard man (1.76 m, 75 kg, 15% fat). In order to enable the user to adjust the simulation to individual anthropometrics, an input option for the values for mass, height, and adiposity of the subject was added. The effects of these variables on body surface area body heat capacity and core-to-skin heat conductance were incorporated in the manner explained below.

Body surface area

Body surface area (A_D) is determined from body mass and height using the equation of DuBois and DuBois (1916). An effect of body surface area in general is the change in heat transfer area. As body surface area can vary substantially between subjects, this is an important effect to be incorporated in the model.

However, body surface area at a certain mass also varies with body fat content. When fat content increases at a fixed mass and height, it will replace heavier tissue types. Thus for the same mass, volume will increase and thus also surface area.

To estimate the magnitude of this effect, we calculated the effect of a large replacement of muscle mass, amounting 10% of body mass, with fat. The density of muscle tissue equals 1.1 kg·l⁻¹, that of fat 0.9 kg·l⁻¹ and that of tissue on average 1.077 kg·l⁻¹ at 10% body fat content. Starting from the fat content of 10% (by weight), the start volume is:

$$\text{start volume} = \frac{75 \text{ kg}}{1.077 \text{ kg·l}^{-1}} = 80.775 \text{ l}$$

The change due to the replacement of 10% of total body weight of muscle mass by fat mass can then be calculated as:

$$\text{volume change for 10\% fat vs muscle} = \frac{-7.5 \text{ kg muscle}}{1.1 \text{ kg·l}^{-1}} + \frac{7.5 \text{ kg fat}}{0.9 \text{ kg·l}^{-1}} = 1.5 \text{ l} \sim 1.9\%$$

The body surface area change related to this 1.9% volume change is dependent of the location where this volume increase takes place. For the most extreme (hypothetical) case, when this volume would be added at the end of an extremity, the surface change would be about 3.5%. For the more likely case, when it would be added to the trunk it would be less than 0.5%. In

view of these minor changes it seems overdone to add this effect of adiposity on A_D to the model.

With increasing A_D, also the area for sweat production will increase. For this reason, the amount of sweat produced by the body is made dependent of A_D, in a linear fashion, using the standard subject (75 kg, 1.83 m) with an A_D of 1.97 m² as a reference. The same approach has been chosen for skin blood flow (more skin area=more flow) and for maximal sweat production and blood flow:

$$\text{sweat or blood flow} = \text{controller output} \cdot \frac{A_D}{1.97}$$

$$\text{maximal sweat production or blood flow} = \text{standard maximum} \cdot \frac{A_D}{1.97}$$

Body heat capacity

Body heat capacity, relevant for determination of the magnitude of the body temperature change at a certain heat storage rate, is mainly determined by body mass. Further, the specific heat of body tissue is of importance. The latter is dependent on body composition. Specific heat of fat is lower than that of other tissues which are mainly water based (blood, muscle). The specific heat of body fat amounts to 2.51 $J \cdot g^{-1}$, whereas that of the other tissues (skin, skeleton, muscle etc combined) is on average 3.65 $J \cdot g^{-1}$ (Stolwijk, 1971; Burton & Edholm, 1955). For the calculation of body temperature changes the following equation for the specific heat of body tissue (c_b) is used:

$$c_b = \left(\frac{\text{fat mass}}{\text{body mass}} \right) \cdot 2.51 + \left(\frac{\text{body mass} - \text{fat mass}}{\text{body mass}} \right) \cdot 3.65 \quad (J \cdot g^{-1})$$

As the distribution of fat over skin and core compartment shows a strong variation between subjects, this specific heat value is taken equal for both segments

Core-to-skin heat conductance

The resistance to heat transport from the body core to the skin is formed by the body shell. This consists of muscle, fat and skin. The muscles are enclosed in the core segment, once they become well perfused as in exercise.
When the shell is vasoconstricted, the heat flow from core to skin is mainly by conductance. When blood flow through these tissues increases, a convective component is added to the heat flow. In the original model, core-to-skin heat conductance is independent of adiposity and only dependent on thermally regulated skin blood flow:

$$\text{Core-skin resistance} = \frac{1}{(5.28 + \text{RAMAN} \cdot 1.163 \cdot \text{skin blood flow})} \quad (m^{2} \cdot {}^{\circ}C \cdot W^{-1})$$

with: 5.28 = skin conductance in the absence of skin blood flow ($W \cdot m^{-2\circ}C^{-1}$)
RAMAN = counter-current heat exchange effectivity
Skin blood flow = skin blood flow in $l \cdot m^{-2} \cdot h^{-1}$ (0.5-90)
1.163 = blood heat capacity in $J \cdot l^{-1\circ}C^{-1}$ divided by 3600 $s \cdot h^{-1}$

This was deemed to be an over simplification in respect to the goal of this study.

Combining data from different sources (Pugh & Edholm, 1955; Nadel et al., 1974; Veicsteinas et al., 1982; Toner et al., 1986; Rennie, 1988), a general model for tissue conductance or resistance can be drawn as shown in Figure 3. This model and the underlying data will be discussed in more detail below.

Most data on core-to-skin conductance were collected in water immersed subjects. Most studies used the concept of "critical water temperature"[2] for the determination of heat transfer properties of the body shell. This implies that in most of these studies, even with increased metabolic rate, skin blood flow will be minimal, or at least relatively low. Thus, it should be kept in mind that the numbers found for resistances of separate heat transfer pathways will pertain to situations with low skin blood perfusion. As skin blood flow is a parallel pathway to that through muscle and fat, values for the latter pathway can thus be successfully derived from the studies mentioned.

Literature data for the body shell insulation range from 0.32 $m^{2\circ}CW^{-1}$ for obese, resting subjects (water; Veicsteinas et al., 1982) to as low as 0.01 $m^{2\circ}CW^{-1}$ for lean, heavily exercising subjects (water; Nadel et al., 1974). Pugh and Edholm (1955) mention an average value for males of 0.093 $m^{2\circ}CW^{-1}$ and of 0.124 $m^{2\circ}CW^{-1}$ for females.

Fat layer: Differences between individuals in shell insulation show a good correlation with subcutaneous fat thickness (Nadel et al., 1974; Veicsteinas et al., 1982; Rennie, 1988). Toner et al. (1986) also observed a relation between total body mass and shell conductance, with big subjects having a lower conductance. As their groups, though having equal fat percentages, differed apart from mass also in total skinfolds thickness, the latter parameter may be the cause of the observed effect and not the actual mass.

Muscle layer: When vasoconstricted, the muscle layer forms a substantial part of the core-to-skin insulation (60-80%, measured in relatively lean subjects; Rennie, 1988). When a person becomes active, the perfusion of the working muscle increases strongly, and the muscle contribution to the shell insulation is extremely reduced (Rennie, 1988). Work rates in

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[2] Critical water temperature: the lowermost limit of water temperature in which insulative regulation of body temperature could occur without increased metabolic heat production.

respective experiments correlated well with reductions in shell insulation (Nadel et al., 1974; Veicsteinas et al., 1982; Rennie, 1988). At high activity levels ($>$ 10 times basal metabolic rate), the shell insulation is between one fifth to one tenth of the maximal (0.05 $m^2 °CW^{-1}$) insulation value (Rennie, 1988).

In the current implementation of core to skin resistance in the model, as mentioned earlier, no relation with body composition is present. Also, metabolic rate has no direct effect on core to skin resistance. In the cold, muscle and skin blood flow are not necessarily related, however. From the literature described above a different representation can be developed following the representation of Figure 3 with the following characteristics:

- increasing work rate and metabolic rate will increase core-to-skin conductance through the increase in muscle blood flow. Since this blood flow is mainly axial (extremities), there is a correlation with radial heat flow, rather then an actual radial convective heat transport;

- skin blood flow in itself will affect core-to-skin conductance through its convective heat transport, which shortcuts the tissue conductive resistances. This was already present in the model;

- the subcutaneous fat layer thickness represents a constant conductive heat resistance.

These points are incorporated in the new representation of core-to-skin conductance in the model using the data from the given literature, with an emphasis on the paper by Rennie et al. (1988). The essentials of the new description are (Figure 3):
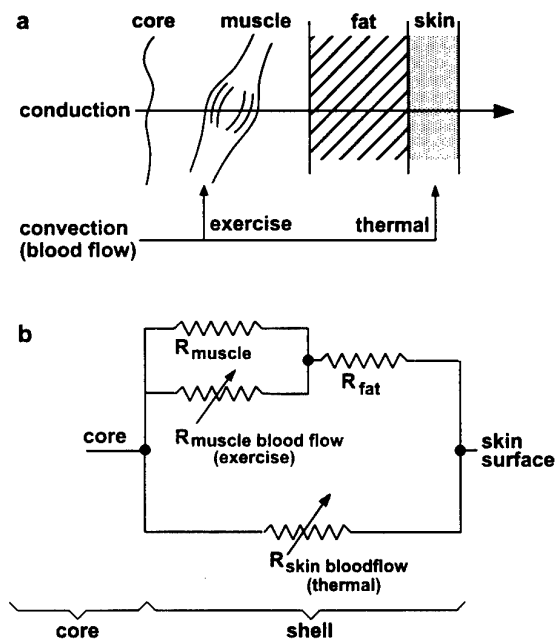

Fig. 3 Schematic representation of the resistors involved in body core-to-skin heat transfer.

$$R_{\text{core to skin}} = \cfrac{1}{\left(\cfrac{1}{R_{\text{skin blood flow}}} + \cfrac{1}{R_{\text{muscle+fat+skin}}}\right)} \quad (\text{m}^{2\cdot\,\circ}\text{C}\cdot\text{W}^{-1})$$

with:

$$R_{\text{muscle+fat+skin}} = R_{\text{fat+skin}} + \cfrac{1}{\left(\cfrac{1}{R_{\text{muscle, conductive}}} + \cfrac{1}{R_{\text{muscle blood flow}}}\right)} \quad (\text{m}^{2\cdot\,\circ}\text{C}\cdot\text{W}^{-1})$$

As muscle blood flow as such is not defined in the model, but this evidently is related to metabolic rate, this factor can be represented as a function of metabolic rate. Skin blood flow is already a parameter in the model, incorporated as a function of body temperature. The other parameters were deducted from the data in the mentioned papers, with the assumption that due to the experimentation in water at critical water temperature, the tissue insulation was maximal and skin blood flow, even during exercise, was minimal. The equations for the three components of core to skin resistance put into the model then read:

$$R_{\text{skin blood flow}} = \cfrac{1}{(1.167\cdot\text{RAMAN}\cdot\text{skin blood flow})} \quad (\text{m}^{2\cdot\,\circ}\text{C}\cdot\text{W}^{-1})$$

$$R_{\text{muscle}} = \cfrac{0.05}{1 + \left(\cfrac{\text{metabolic rate} - 65}{130}\right)} \quad (\text{m}^{2\cdot\,\circ}\text{C}\cdot\text{W}^{-1})$$

$$R_{\text{fat+skin}} = 0.018\cdot\text{Thickness}_{\text{fat+skin layer}} \quad (\text{m}^{2\cdot\,\circ}\text{C}\cdot\text{W}^{-1})$$

The thickness of skin and fat is readily available to model users when they apply the common method of measuring skinfolds thicknesses (preferably an average of > 5 sites) for adiposity assessment.

$$\text{Thickness}_{\text{fat+skin layer}} = 0.5\cdot\text{mean skinfold thickness} \quad (\text{mm})$$

If the skinfolds thickness is not available, but instead only the body fat percentage is known, the mean skinfolds thickness has to be derived from this. Body fat percentage, as measured by under water weighing, would imply conversion problems since Davies et al. (1986) showed that a wide variation exists between subjects in the distribution of total body fat over the subcutaneous fat layer and the internal fat compartments.

Jackson and Pollock (1985) gave relations between % body fat, body density and skinfolds thickness (seven point average):

$$\text{body fat} = \left[\cfrac{4.95}{D_{body}} - 4.50\right]\cdot100 \quad (\%)$$

$$D_{body,\ male} = 1.112 - 10^{-6} \cdot (434.99 \cdot SF + 0.55 \cdot SF^2 - 288.26 \cdot age) \quad (kg \cdot l^{-1})$$

$$D_{body,\ female} = 1.097 - 10^{-6} \cdot (469.71 \cdot SF + 0.56 \cdot SF^2 - 128.28 \cdot age) \quad (kg \cdot l^{-1})$$

with:
D_{body} = average density of body tissue
SF = sum of seven skinfolds.

These equations can be used in an inverse way to retrieve skinfolds thickness from % body fat.

For the model, using gender, age and the body fat percentage as input, the sum of seven skinfolds (SF) was derived with these equations, and from this SF, the average superficial fat+skin layer thickness was calculated:

3.2   Gender and age

The gender and the age of the subjects will not be introduced in the model as factors directly affecting thermoregulation. Enough evidence is present in literature (for a discussion see Havenith, 1985; Havenith et al., 1990, 1995a) showing that the main influence of age and gender in exercise heat exposure really acts through concomitant differences in aerobic power, fat content, mass and A_D. Optionally, the user may be given the choice to select gender and age for an individual subject, but in the model this would be translated in a change in aerobic power, fat content, mass etc. based on epidemiological data of differences in these parameters between genders and ages.

3.3   Sweating control

Individual differences in sweat production can be quite large. This is in part related to large differences in sweat gland size ($1.35-7.3 \cdot 10^3$ mm^3; Sato & Sato, 1983) and distribution and in sweat production per gland (Hertzman, 1957; Kuno, 1956). When heat stress increases, total sweat output differences are reduced, however (Hertzman, 1973). For the modelling of individual differences in sweat output, major parameters are the training level of the subject (Henane et al., 1977; Nadel et al., 1974; Gisolfi, 1984) as well as by the level of acclimatization (Avellini & Kamon, 1980; Candas et al., 1980; Candas, 1980; Davies, 1981; Kobayashi et al., 1980, Mitchell et al., 1976). Most textbooks follow the relations between training, acclimation and sweat rate as presented by Nadel et al. (1974).

Table I Overview of data relating to the effect of training and acclimation on the sweat rate-T_{co} relation. *Data of Roberts et al. (1977) are recalculated with exclusion of an apparent outlier.

source	n	condition	change in T_{co} rest (°C)	$\dot{V}O_{2\,max}$ subjects (ml·kg⁻¹·min⁻¹)	change in $\dot{V}O_{2\,max}$	sweat threshold (°C)	sweat gain	max. sweat rate (g·h⁻¹)
Henane & Bittel, 1975	12	rest + heat	-0.2	?		↓ -0.27	↑(in 50% subjects)	
Wyndham, 1967	13 /353	exercise + heat		apprentice miners versus miners		↓ -0.5 (between groups)		520→800 (+54%)
Kobayashi, 1980						↓		
Nadel et al., 1974	6	exercise training (75% $\dot{V}O_{2\,max}$) heat; exercise (50% $\dot{V}O_{2\,max}$)+ heat		38.1→44.7; ?; ?	+17%; 0?; +17%?	↓ -0.1; ↓ -0.2; ↓ -0.3	+67%; +0%; +67%	
Henane & Valatx, 1973	9	rest + heat				↓	=	
Henane et al., 1977	3	exercise (25/110% $\dot{V}O_{2\,max}$ cycles)	-0.4	40.9→48.3	+18%	↓ -0.1~-0.4	+60%	
Shvartz 1979	10	exercise (50% $\dot{V}O_{2\,max}$) + heat		41	n.s.	↓ -0.49		
Gonzalez, 1974	6	low exercise (25% $\dot{V}O_{2\,max}$) + heat		50.3	n.s.	↓ -0.5	+47%	
Roberts et al., 1977*	8	exercise training (75% $\dot{V}O_{2\,max}$) heat only; exercise (50% $\dot{V}O_{2\,max}$) + heat		42.7→47.7; 47.7→46.9; 42.7→46.9	+12%; -1.7%; +10%	↓ -0.1; ↓ -0.12; ↓ -0.22	+36%; +14%; +54%	
Heany, 1994	10	heat + low exercise (33% $\dot{V}O_{2\,max}$)		48	n.s.			+25% (at lower T_{co})
Havenith & Van Middendorp, 1986	4	heat+low exercise (constant strain)		54	n.s.	↓ -0.25	=	

They suggest that the relationship between sweat rate and core temperature is linear (at constant T_{sk}), and that training increases the gain of this relationship and acclimation decreases the core temperature threshold value. The simplicity of this theory is appealing, but there are two points which justify closer scrutiny of its basis. The presented "typical" graphs always are from single subjects, and responses from different individuals within a group were often inconsistent. Also, the relations are obtained during rapid warming within 10–20 min. A possible time constant of the system, or time lag between the measured esophageal and the actual "core" temperature would have a strong impact on the observed relation (Havenith, 1985). Therefore we chose to reevaluate the literature on the subject of training and acclimation effects on the sweat rate-T_{co} relation. An overview of the findings from which also quantitative data could be obtained is presented in Table I. The presented sweating threshold values are defined as values at which sweat evaporation was found to increase. This is in contrast to some literature, which report extrapolated thresholds. They extrapolate the sweat rate-T_{co} relation back to the point of zero central drive as suggested by Nadel et al. (1974). The latter threshold is the point where the gland is supposed to be activated. The difference between the two definitions is the amount of sweat which has to be produced before it actually reaches the skin surface and evaporates. In the duct, part of the sweat is absorbed. If the zero central drive definition would have to be used for modelling, knowledge of the amount of sweating needed for appearance of sweat at the skin surface would be necessary (sweat duct volume, sweat duct resorption). Insufficient data are yet available for that purpose.

Training versus acclimation

Several studies have separated the actual training effect from that of heat acclimation alone or the combination of heat and training. Comparison of quantitative effects is difficult, as the size of effects is strongly related to the methods used. For training effects e.g., exercise intensities above $50\% \dot{V}O_{2\,max}$ are needed (Pandolf, 1979). In many experiments these were not always warranted (Shvartz et al., 1973; Strydom & Williams, 1969), as aerobic power levels increase in the course of the experiment due to the training. In experiments using a constant absolute training intensity the continuously improving aerobic power will result in a continuous decrease in relative load for the subject.

A problem with the interpretation of data for the "maximal sweat rate" is that usually heat stress tests before and after the treatment (i.e. acclimation) were identical, and thus the treatment may have resulted in a reduced strain in the second test. Thus though equal or even lower sweat rates may be observed after treatment, actual maximal sweat capacity may have increased.

Considering the results summarized in Table I, it seems that heat, exercise and the combination of heat and exercise all have an effect on the threshold for sweat appearance at the skin surface. Threshold shifts due to exercise training alone (changes in $\dot{V}O_{2\,max}$ of 12–17%) range between 0.1 and 0.4°C, but considering the numbers of subjects used in different studies, a shift of 0.1°C seems the best consensus. Heat acclimation following exercise training (Nadel

et al., 1974; Roberts et al., 1977) produces an additional set point shift of 0.12 to 0.2°C. The total threshold reduction of heat+training amounts to 0.22 to 0.5°C. Threshold shifts due to heat acclimation alone are only available from Henane and Bittel (1975) and amount 0.27°C. Low exercise during heat acclimation results in a shift of approximately 0.25 to 0.5°C (Gonzalez, 1974; Havenith & Van Middendorp, 1986), which is not substantially less then heat+high exercise. In general, the main shift in threshold seems to be caused by the heat exposure.

With respect to the change in gain of the sweat rate-T_{co} relation, observations (Table I) range from 36 to 67% increase for exercise training, from 0 to 14% for a subsequent heat with exercise regime and from 54 to 67% for the total of heat + exercise training acclimation. Heat alone (Henane and Bittel, 1975) results in increased gain in part of the subjects, but an average number could not be obtained from the data. Heat+low exercise (Gonzalez, 1974; Havenith & Van Middendorp, 1986), results in 0 to 47% gain increase. Thus training seems the major factor in the gain increase, but heat by itself also has an effect.

The aerobic power level of the subjects used in the training experiments of table I ranged between 38.1 and 42.7 ml·kg^{-1}·min^{-1} before training. Compared to a normative reference (Morrow et al., 1995), these subjects can be characterized as below average to average. The level increased after training to average/above average. Over all experiments, the starting aerobic power level of all subjects ranged from below average to good.
When considering the studied effects over a larger population, one may expect a wider distribution in aerobic power levels, and thus the differences in associated thermoregulatory responses are likely to be larger than observed here.

For modelling purposes, training and acclimation need an operational definition, which will be considered below:

No quantitative data are available on the effect of humid versus dry heat acclimatization on thresholds and gains of the sweat system. Several reports have shown that acclimatization to one type of climate is also beneficial for the other (Bean & Eichna, 1943; Eichna et al., 1945; Griefahn, 1994; Griefahn et al., 1992). For the model, no distinction between dry and humid heat acclimation will be made.
Further, no quantitative data are available on the differences between acclimation to different stress levels (e.g. 30°C and 40°C). The higher the acclimatization load, the higher the expected change in thermoregulatory stability is expected to be. However, as at present this cannot be quantified, it was chosen to operationalize acclimatization in the model as a simple parameter: the number of acclimation days (exposures to a stressful climate (WBGT > 30°C) which increased body core temperature substantially) without further distinction between climate type and heat load level. The shape of the relation (acclimation effect versus number of acclimation days) was adapted from Givoni and Goldman (1973), and Neale et al. (1996):

$$\text{acclimatization} = 1 - e^{-0.3 \cdot \text{number of acclimation days}}$$

The magnitude of the pure acclimation effect, separated from the training effect was derived from the data discussed above.

For a training effect on the regulation characteristics of sweating and skin blood flow to be present, an actual increase in $\dot{V}O_{2\,max}$ has to be observed. Thus the absolute value of $\dot{V}O_{2\,max}$ by itself (a sum of genetic and training influences) does not have to be a valid parameter for the "acclimation like" effects of exercise training. However, a strong correlation of $\dot{V}O_{2\,max}$ with heat tolerance has been observed (Pandolf, 1977, 1979) and it seems fair to use $\dot{V}O_{2\,max}$ (expressed in $ml \cdot kg^{-1} \cdot min^{-1}$ for separation from the mass effect) as indicator for training and aerobic power induced effects on the sweat rate-T_{co} relation.

As mentioned above, the effects described in Table I are for a limited $\dot{V}O_{2\,max}$ range. Thus the model will use this range as a scaling factor for changes outside this range of $\dot{V}O_{2\,max}$ values. In absence of data on the relation between the range of $\dot{V}O_{2\,max}$ and observed effects, the scaling factor is chosen as linear.

The practical implementation of the effects of aerobic power and heat acclimation on the model's sweat control function is illustrated in Figure 4, and can be described as follows: The factor training was implemented as being defined in terms of a range in $\dot{V}O_{2\,max}$, expressed in $ml \cdot kg^{-1} \cdot min^{-1}$. This can be entered directly, or as derived from absolute $\dot{V}O_{2\,max}$ ($l \cdot min^{-1}$). The range used for having an effect on the sweat rate-T_{co} relation is arbitrarily chosen to be 20–60 $ml \cdot kg^{-1} \cdot min^{-1}$. This covers the fitness ranges 'very poor' to 'good' for young and old subjects (Morrow et al., 1995).
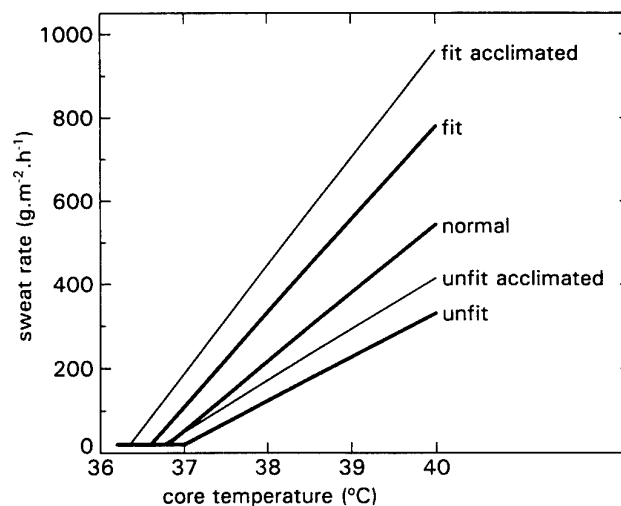

Fig. 4 Graphical representation of the influence of aerobic power and acclimation on the control function for skin blood flow, assuming a constant skin temperature.

For the set point shift related to $\dot{V}O_{2\,max}$, from Table I a number of $0.1°C$ was derived for a $10\ ml\cdot kg^{-1}\cdot min^{-1}$ range, which for the full range $(40\ ml\cdot kg^{-1}\cdot min^{-1})$ is extended to $0.4°C$. For the gain change, the increase seen in Table I of $36-67\%$ for the $10\ ml\cdot kg^{-1}\cdot min^{-1}$ range was extended to 200% for the full range (unfit to fit). Taking the aerobic power average of 40 $ml\cdot kg^{-1}\cdot min^{-1}$ as a reference (18-45 year average; Morrow et al., 1995) this leads to the following training and acclimation based adjustments in the governing equations, which modify set point and gain:

$$\text{fit} = VO_{2\,max}/kg - 40 \qquad (20 < VO_{2\,max}/kg < 60)$$

$$\text{acclim} = 1 - e^{-0.3\,\cdot\,\text{number of acclimatization days}} \qquad (0 \leq \text{number of accl. days} \leq 14)$$

$$\text{setpointnew} = \text{setpoint} - \left(\frac{\text{fit}}{10}\right)\cdot 0.1 - \text{acclim}\cdot 0.25 \qquad (°C)$$

$$\text{gainnew} = \text{gain}\cdot\left[1 + \frac{\text{fit}}{20}\cdot 0.35\right]\cdot[1 + \text{acclim}\cdot 0.15] \qquad (g\cdot m^{-2}\cdot h^{-1}\cdot °C^{-1})$$

For maximal sweat rate (MSR), a difference of 100% between unfit, unacclimatized, and fit, acclimatized was arbitrarily chosen, mainly based on data from metacholine injection studies (Inoue et al., 1997) and studies comparing medium fit unacclimatized subjects with fit acclimatized (Wyndham, 1967, Table I).

$$\text{MSR} = \text{maximum for unacclimatized 40 } ml\cdot kg^{-1}\cdot min^{-1} \text{ person}$$

$$\text{MSR}' = \text{MSR}\cdot\left[1 + \left(\frac{\text{fit}}{20}\right)\cdot 0.25 + \text{acclim}\cdot 0.25\right] \qquad (g\cdot m^{-2}\cdot h^{-1})$$

3.4 Skin blood flow

The regulation of skin blood flow has been studied through skin blood flow in the extremities (plethysmographic techniques) and through core-to-skin conductivity. Extremity blood flow is regarded as indicative/representative for total body skin blood flow, and provides a more direct measure than core-to-skin conductivity does.

Data on the effect of training and acclimation on skin blood flow are limited, and often conflicting. Acclimation results in a reduced core temperature threshold for forearm, hand, chest and ear vasodilation (Fox et al., 1963; Roberts et al., 1977; Gonzalez et al., 1974). Also maximal conductance measured at the chest increases (Gonzalez et al., 1974). Besides increased vasodilation (Wood & Bass, 1960), also venoconstrictor tone increases in the first days of acclimation. Comparisons before and after acclimation do not show changes in skin blood flow, however. This may be due to reduced strain after acclimation, reducing the

necessity of high blood flows at equal stress. Also, venous tone is strongly affected by non-thermal influences.

Wyndham (1951), studied the effect of acclimation (with exercise) during extreme heat exposure. With this maximal stimulus, forearm blood flow increased from 14 to 26 ml·100ml^{-1}·min^{-1}. Roberts et al. (1977) provided quantitative data on set point and gain of the forearm blood flow-T_{co} relation. They observed a significant reduction in threshold for vasodilation of 0.2°C by exercise training ($\dot{V}O_{2\,max}$/kg: 42.7→47.7 ml·kg^{-1}·min^{-1}) and another reduction by 0.26°C by successive exercise+heat acclimation. The change in gain was less consistent. Exercise training resulted in an average gain increase of 1.3 ml·100ml^{-1}·min^{-1}°C^{-1} and subsequent acclimation by heat and exercise in a reduction with 0.8 ml·100ml^{-1}·min^{-1}°C^{-1}. However, the validity of these gain changes for a generalized model is questionable, as different subjects showed very different reactions (4 out of 6 subjects showed the gain increase with training, 2 a decrease; 6 out of 8 the decrease with heat acclimation against 2 with an increase and the total of training with subsequent acclimation showed only 2 out of 6 with an increase and 4 with a decreased gain).

The maximal value of skin blood flow in relation to aerobic power has received little attention in literature. As in the heat a competition exists between blood flow for supply of nutrients and oxygen to muscles and skin blood flow for core-to-skin heat transport, it is likely that a high maximal cardiac output is a good indicator for the ability to produce and maintain a high skin blood flow. Maximal cardiac output is strongly related to $\dot{V}O_{2\,max}$ (Ekblom, 1969 in Åstrand & Rodahl, 1970). Thus it seems reasonable to relate maximal skin blood flow in the model to the parameter for aerobic power. Acclimation will have an effect on maximal skin blood flow due to its stabilizing effect on circulation. The size of this effect is supposed to be smaller than that of aerobic power, however.

In the original model the basal skin blood flow rate is 6.3 l·m^{-2}·h^{-1} with a maximum of 90 l·m^{-2}·h^{-1}. Skin blood flows measured with plethysmographic techniques are around 1 ml·100ml^{-1}·min^{-1} at rest and on average 15 ml·100ml^{-1} ·min^{-1} at maximum. This is the same ratio. The maximal skin blood flow between subjects of different aerobic power levels usually ranges between 10 and 20 ml·100ml^{-1}·min^{-1} (Havenith, 1995). Thus, translated to model units the maximum should range from 60 to 120 l·m^{-2} ·h^{-1} for different aerobic power levels, with a mean of 90 l·m^{-2}·h^{-1} at an average aerobic power.

For the model, equations graphically represented in Figure 5 were used:
For the set point shift, considering the limited amount of data, an analogy with the set point shift due to training for sweating was chosen. Again, a person with a $\dot{V}O_{2\,max}$/kg of 40 ml·min^{-1}·kg^{-1} was taken as reference. For acclimation a shift of 0.25 was taken.

$$\text{fit} = VO_{2\,max}/kg - 40 \qquad (20 < VO_{2\,max}/kg < 60)$$

$$acclim = 1 - e^{-0.3 \cdot number\ of\ acclimatization\ days} \qquad (0 \leq number\ of\ accl.\ days \leq 14)$$

$$\text{setpoint new} = \text{setpoint} - \left(\frac{\text{fit}}{10}\right) \cdot 0.1 - \text{acclim} \cdot 0.25 \quad (^\circ\text{C})$$
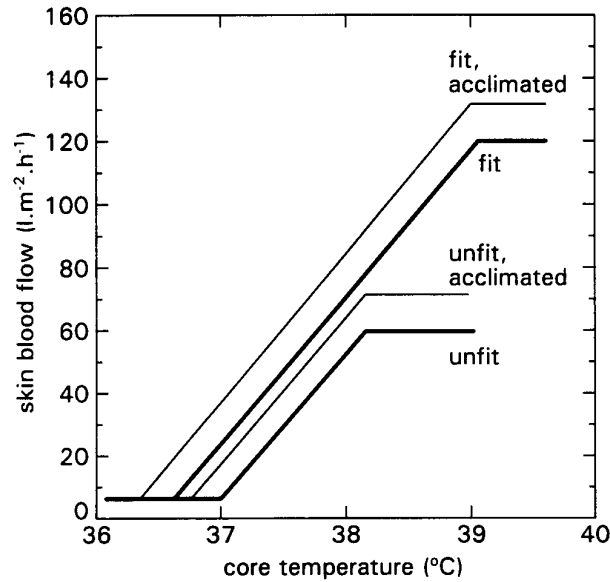

Fig. 5 Graphical representation of the influence of aerobic power level and acclimation on the control function for skin blood flow, assuming a constant skin temperature.

For the gain, no effect was introduced due to the inconsistency in the data.

For maximal skin blood flow (MSBF) the effects of aerobic power and acclimation were formulated as follows:

$$\text{MSBF} = \text{maximum for unacclimatized } 40 \text{ ml·kg}^{-1}\text{·min}^{-1} \text{ person}$$

$$\text{MSBF}' = \text{MSBF} \cdot \left[1 + \left(\frac{\text{fit}}{60}\right) + \text{acclim} \cdot 0.15\right] \quad (\text{ml·100 ml}^{-1}\text{·min}^{-1})$$

3.5 Mean body temperature calculation

In the model 2 nodes are treated separately: core and skin. The core compartment is defined as having a regulated, uniform temperature which is defended against external influences. The shell is used as an intermediate layer between core and environment, which temperature is less uniform, and which temperature defence is subordinate to that of the core. The size of these nodes is not constant. When a subject is highly vasoconstricted, perfusion of the skin and the extremities will be minimal. The body shell will cool down and become relatively larger in size than in a vasodilated state. For calculations of body heat content, mean body

temperature, or changes in node temperature with a certain heat influx, the current model uses a parameter α to present the relative contribution of the shell (skin) node to the total body:

$$T_{body} = (1-\alpha) \cdot T_{re} + \alpha \cdot T_{sk} \quad (^{\circ}C)$$

The size of α is in the original model dependent on skin blood flow:

$$\alpha = 0.042 + \frac{0.745}{(\text{skin blood flow} + 0.59)} \quad (\text{n.d.})$$

The value of α in neutral to hot conditions varies in simulations between 0.15 to 0.05. These values are very low, compared to literature, as can be seen in Figure 6. In this figure, the relative size of the core compartment (1-α) is presented, since that is more common in literature. The low α in the model also has consequences for the model's behaviour. If e.g. condensation on the skin takes place, T_{sk} rises disproportionally due to the liberation of the heat of condensation in a very thin skin layer. One reason behind the calculation resulting in these low α values in the original model may be that α not only determines the weighing of skin versus core for the body heat content calculation, but it also determines the weighing for the sensitivity of sweating and skin blood flow to temperature changes in the skin versus changes in the core compartment. Most literature actually suggests a sensitivity for skin temperature less than one tenth of that of core temperature (Stolwijk, 1971; Stolwijk & Hardy, 1977), suggesting α values below 0.1.
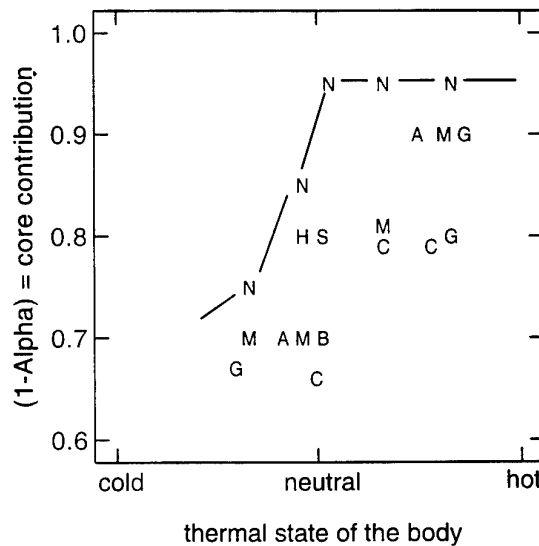

Fig. 6 Proposed values for the relative size of the core compartment (1-α) from different sources are presented, including the α-values for the original model (line). N=Gagge et al., 1986; G=Gagge & Nishi, 1977; B=Burton & Edholm, 1955; H=Hardy & DuBois, 1938a,b; S=Stolwijk & Hardy, 1966a,b; C=Colin et al., 1971; A=ASHRAE; M=Malchaire, 1996.

For the model, these two meanings of α will be separated into a fixed sensitivity for core versus skin and a variable α value for the determination of the shell size. The data in Figure 6 suggest that the minimal value for α should be 0.1, and the total range from cold to hot should be between 0.35 and 0.1.

As α is dependent on the actual core-to-skin resistance and as the effect of adiposity and muscle blood flow on core-to-skin resistance are now incorporated in the model, it seems logical to have α incorporated in the model as a function of total core-to-skin resistance. The following equation provides a good representation of the α-T_{co} relation:

$$\alpha = 0.08 + 2 \cdot \text{Resistance}_{core-skin} \quad (0.1 < \alpha < 0.35)$$

The effect of using this equation is presented in Figure 7. There 1-α is shown in relation to T_{re} for both the original and the new model, assuming a constant skin temperature of $34°C$. The latter causes the limitation of 1-α to $\pm$ 0.75, as a lower 1-α will only be achieved at lower skin temperatures. The change between the old and the new model is a combined effect of the new representation of α itself and the effect of the changed control curve for skin blood flow on the α-T_{re} relation.
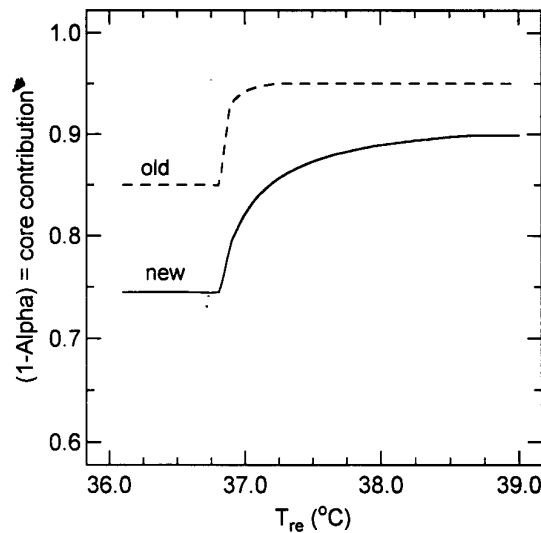

Fig. 7 Effect of altered representation of the core and skin compartment contributions (1-α and α) to the total body compartment on the relation between T_{co} and α, assuming a constant skin temperature. Old=original model, new=present model.

4   VALIDATION

With all the listed changes incorporated in the model, the question needs to be answered whether the changes actually lead to an improvement of the prediction results. For this purpose, the model's prediction for individual heat stress responses was compared to data sets which have not been used in the design of the model, and which are therefore truly independent for validation purposes. The validation was performed using data obtained by Havenith et al. (1990, 1995a,b, 1996) in a cool (CO; 20°C, 50%), warm humid (WH; 35°C, 80%) and a hot dry (HD; 45°C, 20%) climate, exercising either at fixed workloads (ABS; 60 Watt) or loads relative (REL, 25/45%) to the individual maximum $\dot{V}o_2$. For each subject and condition, a separate simulation run with the actual data for climate, work load, metabolic rate, $\dot{V}o_{2\,max}$, body mass, body fat content, height, and acclimatization status was performed. Acclimatization was set to zero for all subjects, and was thus not a part of this evaluation. The approach used for heat acclimatization/acclimation in the current model has been evaluated before by Neale et al. (1996), with good results.

The validation with the data sets mentioned resulted in 181 simulation runs. The final values for body core temperature, skin temperature and body heat storage were subsequently used for evaluation of the model's performance.

In Figure 8, the relation between the calculated and observed heat storage is presented graphically. From this graph, it can be concluded that the model does not always predict around the line of identity. In some conditions the calculations systematically overestimate heat storage, in others it underestimates it. Over all conditions, the average prediction is very close to the average measured value. For body core temperature for instance, the difference is only 0.07°C. The systematic deviation is not the topic of this study, however. That is the improvement due to individualization. To look at this specific performance of the model, for each experimental condition the simulated values were compared to the real values for these variables. The correlations between the two were determined. The improvement of the model's performance due to incorporation of individual characteristics was then defined as the correlation coefficient and the squared correlation coefficient ($\approx$ explained variance) between predicted and real values of T_{co}, T_{sk} and heat storage. For the non-individualized model, the correlation and the explained variance will be 0 by definition, as an equal prediction is given in that case for all subjects. The results are presented in Table II. This Table shows that the predictive value for the model varies strongly between conditions. Calculating the explained variance for body heat storage from the presented numbers (r^2) this explained variance due to the individualization of the model amounts to 77% over all conditions, it varies between 4% (condition CO/REL) and 53% (WH/REL).
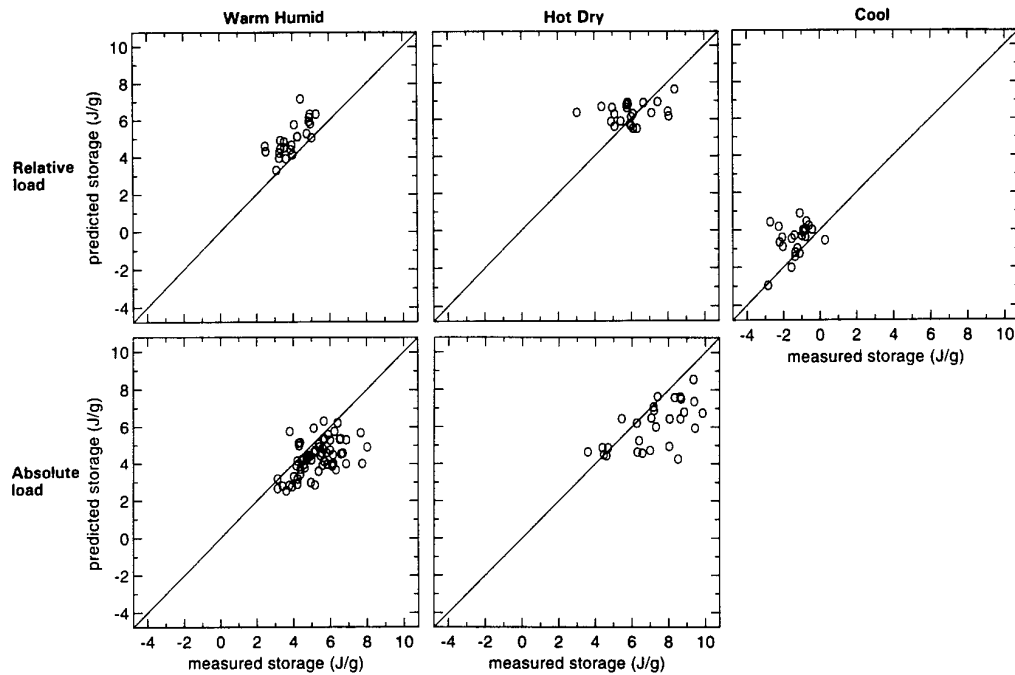
Fig. 8 Relation between predicted and real body heat storage (J·g^{-1} body mass) for the separate conditions of the experiments.

Table II Standard deviation (sd) of original data and correlation coefficients (r) of relation between simulated and real physiological responses attributed to the incorporation of individual characteristics in the model.

condition	number of cases	T_{re}		T_{sk}		body heat storage	
		SD (°C)	r	SD (°C)	r	SD (J·g^{-1})	r
CO/REL	24	0.29	-0.09	1.00	0.47	1.04	0.19
WH/REL	24	0.23	0.67	0.34	0.73	0.81	0.73
HD/REL	24	0.36	0.24	0.74	0.33	1.29	0.26
WH/ABS	82	0.37	0.57	0.40	0.04	1.15	0.52
HD/ABS	29	0.45	0.65	0.75	0.26	1.65	0.64
overall	183	0.45	0.63	2.70	0.95	2.84	0.88

Thus for several conditions (CO/REL and HD/REL) the models predictive capacity for effects of individual differences is low (< 10%), and for others (WH/REL, WH/ABS, HD/ABS) it is significant (27–53%).

In the papers, in which the used data were described, the data were analysed for the influence of individual characteristics on the heat stress response by multiple regression analysis. It is interesting, considering these data sets, to compare the model's predictive capacity to the

predictive power of the multiple regression models, which directly link the subject character-istics with the response. This comparison for body core temperature is presented in Table III.

Table III The correlation coefficients of the predicted T_{re} of the computer model and the real T_{re} data, and the correlation of the estimated T_{re} 's by the multiple regression model and the real data. The last column gives the performance of the independent, analytical, computer model as a percentage of the performance of the empirical regression model, the latter based on the data sets used.

condition	number of cases	correlation of model and real T_{re}		ratio r^2 simulation regression (%)
		r multiple regression model	r computer simulation model	
CO/REL	24	0.50	-0.09	-
WH/REL	24	0.71	0.67	89
HD/REL	24	0.31	0.24	60
WH/ABS	82	0.77	0.57	54
HD/ABS	29	0.71	0.65	84

As the empirical regression model was derived from these specific data sets and from the characteristics of actual participating subjects, the explained variance in these regression models may be considered as the maximum achievable explained variance. Comparison of the two model types shows that except for the cool condition, the (independent!) computer model predicts quite well the variance in the data for T_{re} that could be attributed to individual characteristics (last column in Table III).

The reason for the bad prediction in the cool climate may be due to a too small effect of body fat content on insulation in the model, as in the multiple regression model it was adiposity that had the strongest influence in this condition. The insulative effect of adipose tissue is in the model strongly dependent on skin blood flow. In the model, for most subjects this increases above 30 $l \cdot m^{-2} \cdot h^{-1}$ for the cool condition, which is about one third of the maximum skin blood flow. In the actual experimental data, the forearm blood flow is for most subjects below 3 $ml \cdot 100ml^{-1} \cdot min^{-1}$, which is about one tenth of the maximum. Thus the insulative effect in the cool condition may well be underestimated due to a too high skin blood flow, which may explain the poor predictive effect for that situation.

5   CONCLUSION

The introduction of individual characteristics in a computer simulation model of human thermoregulation contributes significantly to the model's predictive power for individual's heat stress response. The size of the contribution varies with the experimental conditions. For

most conditions it approaches the predictive power of a multiple regression model based on the original data, which may be seen as maximal achievable predictive power from individual characteristics. Nevertheless, still a substantial part of the differences in individual responses remains unexplained.

6 EXAMPLE SIMULATIONS

In the previous paragraphs, the developed computer model was validated against existing data sets. This validation by itself does not provide much information on the versatility of the model in relation to individual characteristics. A good way to provide this information is by presenting examples of model simulations for typical problems encountered in individual heat exposure. This will be done in this section. 6 examples have been selected related to the following topics:

- the difference in response of an "average" male versus an "average" female
- the response of an obese versus a lean person in the heat
- the response of an obese versus a lean person in a cool climate
- the response of a big versus a small subject
- the effect of acclimation and aerobic power on heat tolerance and water loss in chemical protective clothing
- running a marathon; effect of aerobic power, acclimation and running time.

6.1 Man and woman; taking a hike or the bike? (male versus female)

What is the heat strain of an average man or woman who go together on a hiking trip in the heat, on a calm day, both carrying a 10 kg back pack? Can they prepare for that by staying at home and doing the work on an exercise bike with an equal load setting for both in a hot, but well ventilated room?

As described in the previous chapters, the difference in response between males and females on a population bases can be attributed to differences in individual characteristics between the average male and the average female. To see how the model predicts the average responses of the genders, simulations were performed with settings derived from tables on population distributions (Morrow et al., 1995; Daanen, personal communication) as presented in Table IV:

Table IV   Parameter settings for male-female simulations with the individualised computer model.

	male	female
age (y)	30	
mass (kg)	75	65
height (cm)	170	183
adiposity (% fat)	20	25
aerobic power (ml $kg^{-1}min^{-1}$)	42	36
acclimatization (days)	0	
metabolic rate (W m^{-2}) run C	230	257
metabolic rate (W m^{-2}) run M	260	230

The two work load types represent the following situations:

C Cycling on a cycle ergometer at an external work load of 75 Watt, resulting in a metabolic rate of approximately 450 Watts, at an air velocity of 1.7 $m \cdot s^{-1}$. This load is equal for both genders.

M Marching with a load (clothing + back pack) of 10 kg at a speed of 6 $km \cdot h^{-1}$ (air velocity of 1.7 $m \cdot s^{-1}$), no inclination, smooth surface. In this case the metabolic load is dependent on total mass.

To look at the effect of the climate type (i.e. different heat loss pathways), both load types were simulated in a warm humid (WH, 35°C, 80% r.h.) and in a hot dry (HD, 45°C, 20% r.h.) climate. The results for the body core temperatures of these simulations are presented in Figure 9.

While apparently no steady state is reached in the WH climate, this is reached in the HD conditions. For cycling (9a,c), which represents a lower relative load (%$\dot{V}O_{2\,max}$, load/A_D) to the male, he is at a substantial advantage in both climates. In the marching condition (9b,d), the differences between genders are much smaller. In the HD climate the male has a small advantage, but in the WH climate the female is at an advantage, due to the lower absolute heat liberation in the body for the female and despite the limited heat loss.

Thus, the conclusion is that they will have to adjust the ergometer load to a lower load for the female, if they want to produce a comparable strain to marching.
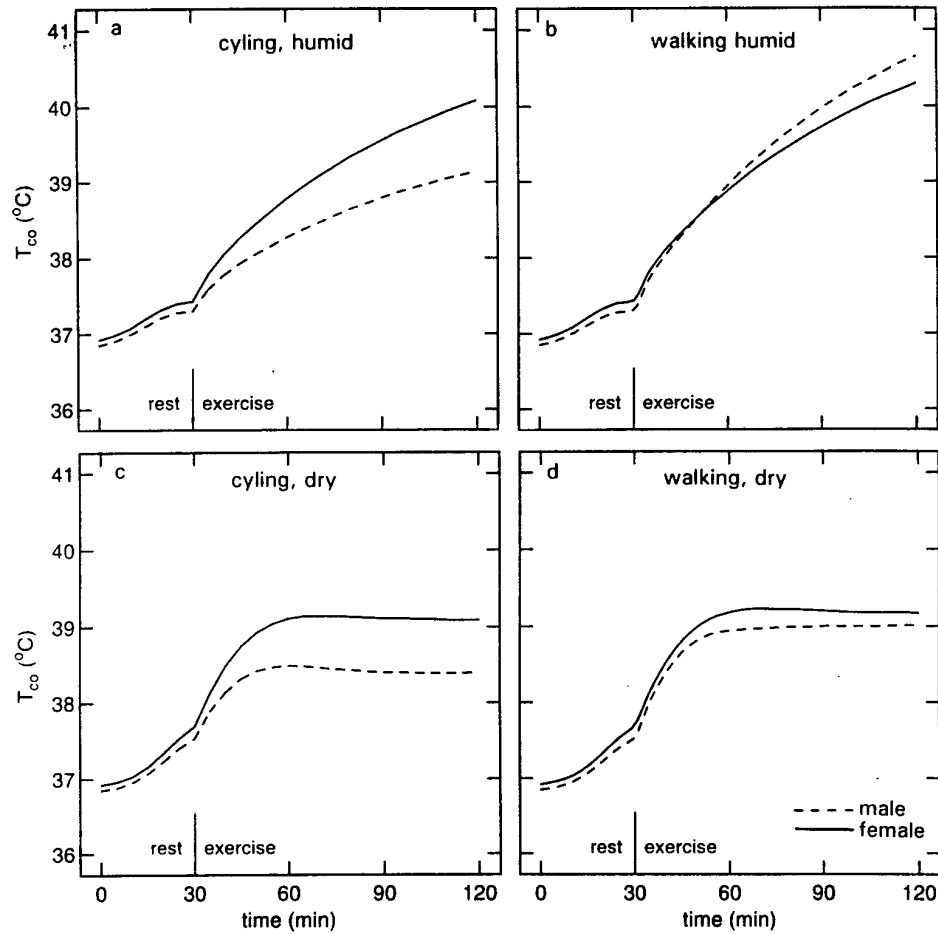
Fig. 9 Results of the simulation runs for the warm humid climate (top) and for the hot dry climate (bottom) with cycling (left) or walking (right) exercise.

6.2 Should we be lean?

A lean and an obese man, dressed in a combat suit, walk (6 km·h⁻¹) together for 60 min after 30 min rest in a WH and a HD climate. They have an equal aerobic power and weight and are both unacclimatized. They differ in body composition: the lean man has 15% fat, the obese 35%. Is the higher body fat content per se, except for all published associated health risks, also a disadvantage in the heat? What about the cold? Does the model show the expected advantage for the obese?

Table V Parameter settings for lean-obese simulations.

	lean	obese
age (y)	30	
mass (kg)	80	
height (cm)	183	
adiposity (% fat)	15	35
aerobic power (ml kg^{-1}min^{-1})	40	
acclimatization (days)	0	
metabolic rate (W m^{-2})	250	

Climates HD and WH (Figure 10a,b) were defined as in example 1. In these climates, only minor differences in the development of body core temperature were observed. Thus nor the difference in body heat capacity between these persons of equal weight, nor the insulating effect of fat had sufficient impact to produce a significant difference in body core temperature response. Overall, the WH climate is more stressful than the HD, though.

In the cool climate (10°C, Figure 11), at rest the lean subject is substantially higher in skin temperature and lower in core temperature (Figure 11a,b). The obese subject not only has a lower heat loss, but is also better able to preserve the heat generated by shivering in this case. The lean subject has to shiver more (Figure 11c) to achieve less.

The skin blood flow (Figure 11d) is minimal for both subjects at rest, but when heat has to be lost during exercise, the obese subject has to send more blood to the skin to get rid of the heat in his core. Though he does increase his skin blood flow fast and substantially, it is for a small period insufficient to prevent an overshoot in T_{co} (Figure 11d,a).

Taking the reaction in the cold and the heat together, being in good shape otherwise, having more fat seems an advantage from the thermoregulatory standpoint. On the other hand, obesity usually correlates negatively with being in a good shape.
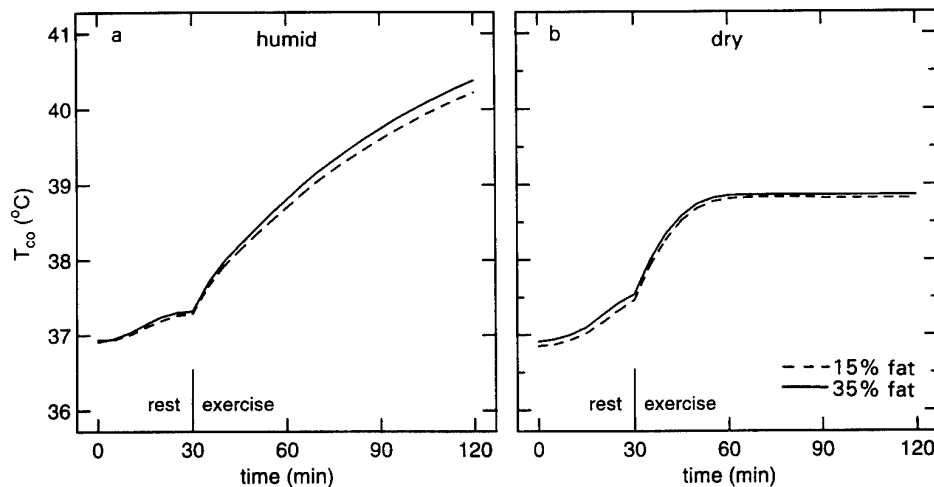

Fig. 10 Effect of obesity on body core temperature response in a warm humid (a) climate and in a hot dry (b) climate.
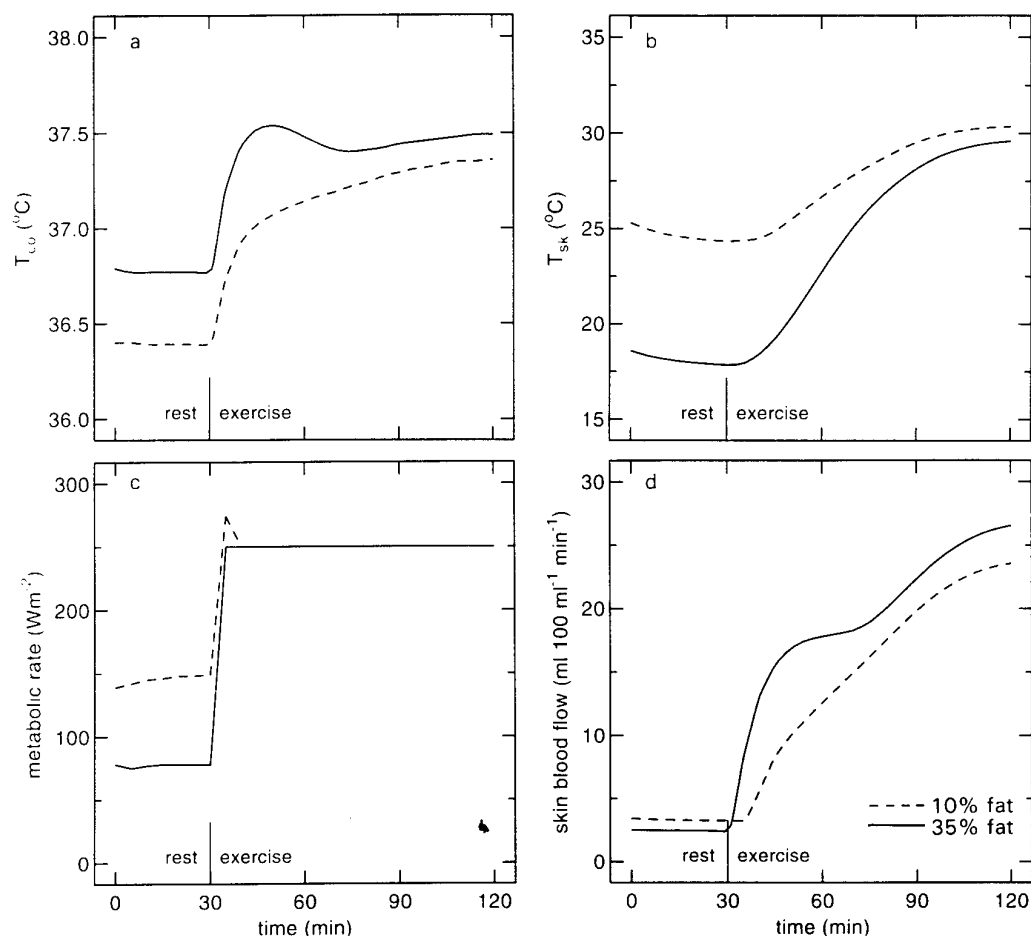
Fig. 11 Effect of obesity on body core- (a) and skin temperature (b), metabolic rate (c) and skin blood flow (d) while resting and exercising in a 10°C environment.

6.3 The advantage of being big

Body weight below 50 kg is considered a contra-indication for work in the heat (Wyndham & Heyns, 1973). In how far can the low mass and low A_D be blamed for that? As a good correlation exists in the population between $\dot{V}O_{2\,max}$ and mass (within a certain range), the small subject usually has a lower work capacity, and will be more stressed when he has to do the same work as his bigger counterpart (see also Figure 9a,c). What if except mass and A_D, all other parameters are equal? This is simulated, using the subject of example 2 with 15% fat and masses of 50 and 80 kg. Both work at the same absolute load. The result for the core temperature prediction is presented in Figure 12.

For both the WH and HD climates, the bigger subject is at an advantage. He has the same heat production as his small colleague, but he has a bigger surface area for heat loss and his mass provides a bigger heat sink. The model thus supports Wyndham and Heyns finding, although there is no special support for setting the lower limit at 50 kg.

Fig. 12 The effect of different masses on body core temperature response in a warm humid (a) and a hot dry (b) climate.

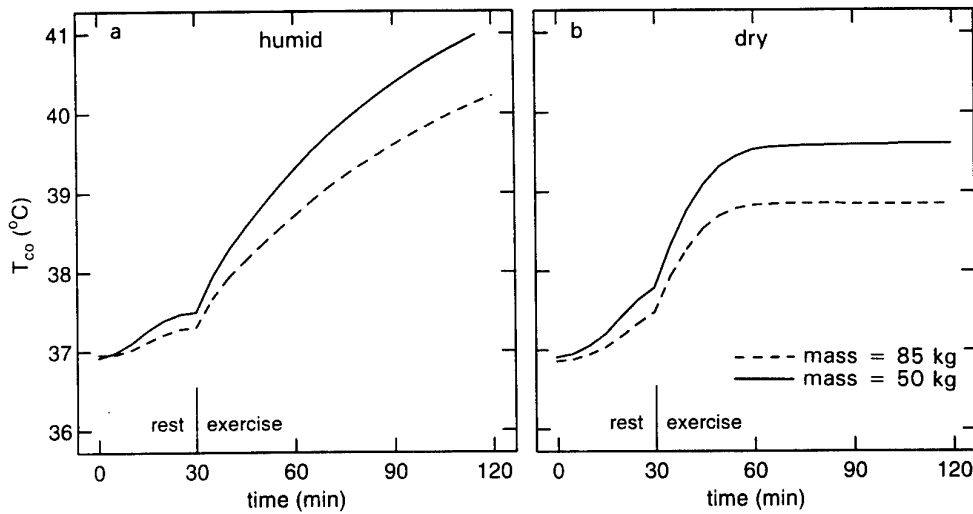## 6.4   Fit for sports! Also fit for work in decontamination suits?

In order to study whether acclimatization and aerobic power are relevant parameters for work in impermeable clothing, two identical subjects (see example 2, lean), except for their aerobic power and their acclimation status, were exposed to a 30°C, 80% humidity environment in a chemical decontamination suit, including respirator and gloves. One subject was set at the moderate fitness, a $Vo_{2\,max}$ of 30 ml·kg^{1}·min^{1} with 0 acclimation days, the other at the high level of 60 ml·kg^{-1}·min^{1} and 15 acclimation days. The responses for core and skin temperature and for accumulated water loss are presented in Figure 13.

As the microclimate in the suits becomes saturated fast with water vapour, the effectivity of sweating is strongly reduced. Evaporative heat loss is limited by the microclimate and not by sweat production. In the saturated microclimate heat loss from the skin is dependent on skin temperature only and not on the subject's acclimation state or his aerobic power. This is reflected in the skin temperature response: after a small difference in the transient state, skin temperatures of both subjects become equal and rise with the same rate (Figure 13b). The same is then expected to happen with core temperature. The rate of rise indeed is identical for both subjects, but due to the lower skin blood flow and the higher relative work load, T_{co} is higher for the "low sweater" (Figure 13a). This lower blood flow necessitates a higher gradient between core and skin to get the same heat transport. Thus the "low sweater" will cross the risk level of T_{co} earlier. The dripping sweat causes in increasing difference in total body water loss between the subjects (Figure 13c) even though the difference in T_{co} stayed equal. Looking at a maximal dehydration level, the "low sweater" would be able to perform longer than the "high sweater". For this type of work the answer to the question which limit will be reached first, the T_{co} limit or the dehydration limit is determined by the work rate and the environmental temperature. Are these such that T_{co} rises slowly (low work, low climatic load), the dehydration will become dominant and the "high sweater" will drop out first. For a

fast rising T_{co}, the "low sweater" will drop out first. Using the model, varying the climate conditions allows the user to analyse the risk for specific situations.
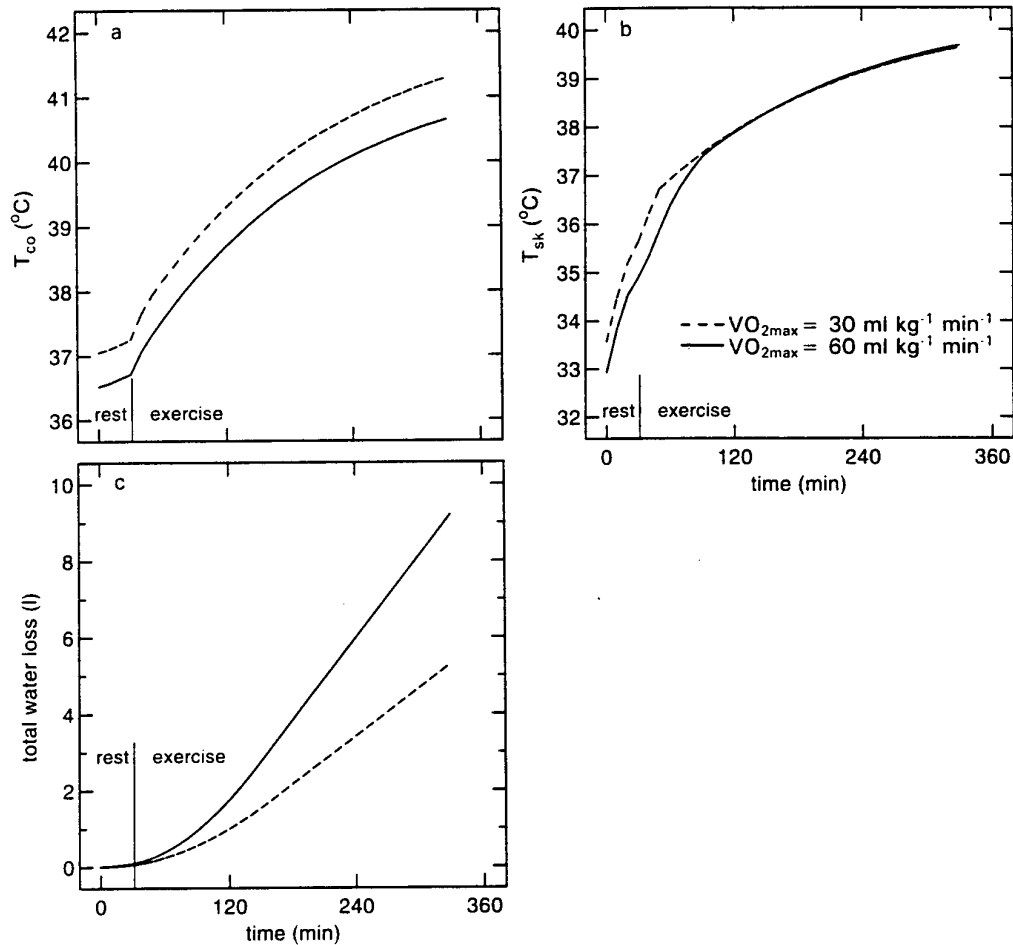
Fig. 13 The effect of aerobic fitness and acclimation on responses of core (a) and skin (b) temperature and of total body water loss (c).

6.5 Marathon time and heat exhaustion risk

Running a marathon in a warm climate is not without risk. In order to look at the risk of overheating, simulation runs were obtained for a top athlete (running scheme time 2:10) with and without heat acclimation, and for a partially acclimated runner with a top time of 2:35. Energy expenditure was derived from the relation between running speed and efficiency for top long distance runners and slower runners as given by Fox and Costill (1972) (Table VI).

Table VI Parameter settings for runners simulations.

	top runner		slower runner
age (y)	30		
mass (kg)	65		
height (cm)	180		
adiposity (% fat)	15		
aerobic power (ml kg^{-1}min^{-1})	85 (model limit 60)		60
acclimatization (days)	0	15	4
running speed (m min^{-1})	325		270
energy cost (ml O$_2$ m^{-1} kg^{-1})	0.17		0.18
work load (W m^{-2})	697		617

The climates used were 30°C, 80% r.h. for the humid condition and 38°C, 20% r.h. for the dry condition. The results for the development of T_{co} are presented in Figure 14a and b. For the dry climate (Figure 14b) both the acclimated top runner as well as the "slow" runner are able to run with a steady state core temperature. As this is around 40°C, it is questionable whether the "slow" runner can actually tolerate this. The top runner probably can, as for most top athletes tolerances to temperatures above 40°C have been observed. For the top runner, the steady state reached is a critical one. If he were unacclimated, he would not be able to achieve sufficient evaporative cooling due to insufficient sweat production, and his temperature would continue to rise, as seen in Figure 14b.

For the humid climate, running a 2:10 marathon is more critical for body temperature (Figure 14a). Here the slow runner again stabilises around 40°C, but the top runner does not reach a steady state. If acclimatized he may be able to finish his run just above 41°C, but unacclimatized a finish in that time is quite unlikely.

To illustrate the effect of running time on T_{co} development, this relation is presented in Figure 14c for a partially acclimatized top-runner. This graph shows that for the chosen subject a steady state in core temperature is possible up to a running time of 2:15. In order to run at that steady state, he has to be able to tolerate a core temperature of up to 40.3°C. Running faster in the simulated climate, results in a continuous increase in T_{co}, and heat exhaustion, before the finish line, will be inevitable. These findings are in line with the risk assessment of the climate (38°C, 20% r.h.) based on the Heat Stress Index, which indicates high heat stress, and on the ACSM guidelines based on WBGT which suggest a moderate heat stress (Vogel et al., 1993).

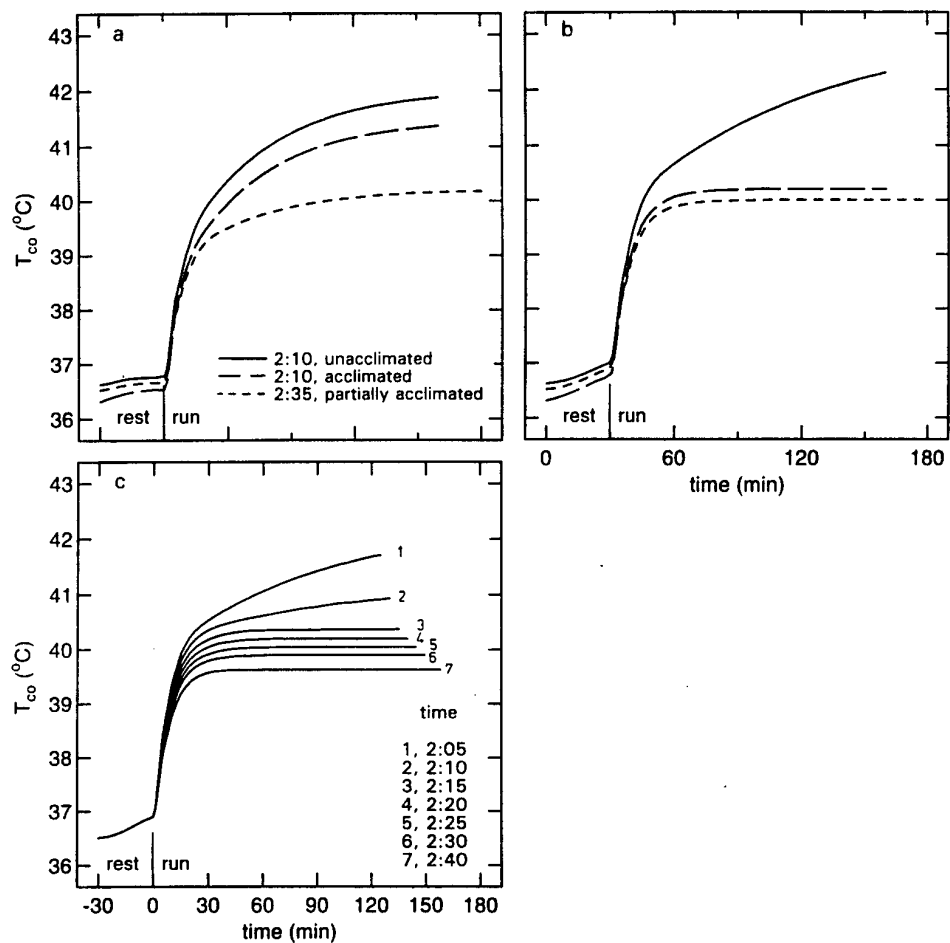The effect of dehydration was not considered in these runs. This is expected to aggravate the risk.

Fig. 14 Body core temperature response while running a marathon in a warm humid (a,c) or a hot dry (b) climate at different speeds.

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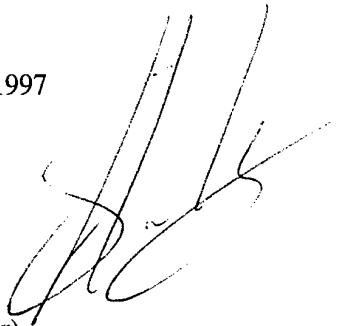
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Soesterberg, 25 June 1997

Drs. G. Havenith
(Author, Project leader)

APPENDIX   Program code, specifically related to individualized model

```fortran
      SUBROUTINE INDIN
C  This subroutine deals with the input of the characteristics,
C  and the calculation of the effects on model parameters.
C
$INCLUDE:'THINDIVO.FOR'
C  changes for initialization of individual differences
      WRITE(*,'(/,'' Give the persons body weight in kg '',
     >       ''(now '',F6.1,''kg):'',\)')W
      CALL GET(W)
      WRITE(*,'(/,'' Give the persons height in m '',
     >       ''(now '',F6.2,''m):'',\)')HEIGHT
      CALL GET(HEIGHT)
      WRITE(*,'(/,'' Give the persons body fat percentage '',
     >       ''(now '',F6.1,''%):'',\)')PFAT
      CALL GET(PFAT)
      WRITE(*,'(/,'' Give the persons age '',
     >       ''(now '',F6.1,''):'',\)')AGE
      CALL GET(AGE)
      WRITE(*,'(/,'' Give the persons gender (M/f)'',\)')
      READ(*,'(A1)')DUM
      IF (DUM.EQ.'f'.OR.DUM.EQ.'F') THEN
            SEX='F'
      ELSE
            SEX='M'
      ENDIF
      WRITE(*,'(/,'' Give the persons $\dot{V}O_{2\,max}$ in ml/kg/min'',/,
     >       '' (standard=40) '',
     >       '' (Enter 999 if you want to enter a relative value '',/,
     >   '' (now '',F6.1,''):'',\)')$\dot{V}O_{2\,max}$
      CALL GET($\dot{V}O_{2\,max}$)
      TRAIN=($\dot{V}O_{2\,max}$-20.)/40.*100.
C  vo2max=40 ml/kg min AT 50% TRAIN;
C  train=100% then 60 ml/kg min
      IF($\dot{V}O_{2\,max}$.GT.100.)THEN
          WRITE(*,'(/,'' Give the persons training level (0-100%;'',
     >             ''(standard=50) '',
     >             ''(now '',F6.1,''%):'',\)')TRAIN
          CALL GET(TRAIN)
          $\dot{V}O_{2\,max}$=20.+TRAIN/100.*40.
      ENDIF
      WRITE(*,'(/,'' Give the number of days the person has acclimated
     >       (0-15; now '',F5.1,''):'',\)')ACCL
      CALL GET(ACCLI)
C  substract first day (see Neale, LUT 25 model)
      ACCL=ACCLI-1
      IF (ACCL.LE.0.) ACCL=0.
C  define acclimation function
      ACCLIM=1.-exp(-0.3*ACCL)
C  calculate derived parameters:
      ADU=W**.425*HEIGHT**.725*.2024
      LBM=.01*(100.-PFAT)*W
C  calculate size factors: LBM and ADU related to standard man
      XLBM=LBM/63.8
      XADU=ADU/1.965
      XW=W/75.
C  calculate specific heat
      HSPEC=.01*(PFAT*2510.+(100.-PFAT)*3650)
C  calculate fat+skin layer thickness from fat percentage and age and gender
```

```fortran
C   BODY DENSITY (siri)
    DENSIT=(4.95/(PFAT/100.+4.5))
C   DETERMINE SSSF = SUM OF SEVEN SKINFOLDS BY ITTERATION
    SSSF=0.
    X=3.
    IF(SEX.EQ.'M') THEN
        DO WHILE (X.GT.DENSIT)
            X=1.112-0.00043499*SSSF+0.00000055*SSSF*SSSF-0.00028826*AGE
            SSSF=SSSF+1.
C       if quadratic function does not reach a minimum, limit SSSF to data domain in reference

            IF(SSSF.GT.280.) THEN
            X=DENSIT
            WRITE(*,'(/,'' DENSITY IN INFINITE LOOP; STOPPED!!; any '',
   >               ''key to continue'',\)')
            READ(*,'(A1)')DUM
            ENDIF
        END DO
    ELSEIF(SEX.EQ.'F') THEN
        DO WHILE (X.GT.DENSIT)
            X=1.097-0.00046971*SSSF+0.00000056*SSSF*SSSF-0.00012828*AGE
            SSSF=SSSF+1.
            IF(SSSF.GT.280.) THEN
                    X=DENSIT
                    WRITE(*,'(/,'' DENSITY IN INFINITE LOOP; STOPPED!!; any'',
   >                   '' key to continue'',\)')
                    READ(*,'(A1)')DUM
            ENDIF
        END DO
    ENDIF
C   THICKNESS SKIN+FAT = 0.5 * SSSF /7.
    FATTH=SSSF/14.
C   calculate effects of training and acclimation on sweat and bloodflow
C   limit aerobic fitness
    IF(VO2 max.GT.60.) THEN
        FIT= 20.
    ELSEIF(VO2 max.LT.20.) THEN
        FIT= -20.
    ELSE
        FIT=VO2 max- 40.
    ENDIF
C   determine effects of acclimation and aerobic power on control functions
C   offsetsweating: effect of training, heat acclim.
    OFFSW= - FIT/10.*0.1 - acclim*0.25
C   GAIN SWEATING:
    GAINSW=(1.+FIT/20.*0.35)*(1.+acclim*0.15)
C   maximal sweat rate:
    XSWMAX=1.+acclim*0.25+FIT/20.*0.25
C   blood flow:
    OFFFL=-FIT/10.*0.1 - acclim*0.25
    GAINFL=1.
    XFLMAX=1.+acclim*0.15+FIT/60.
c
    RETURN
    END



    SUBROUTINE THREG
C
$INCLUDE: 'THINDIVO.FOR'
C   calculates physiological control according to a modified Gagge approach;
C   includes, individualization of subject response (Havenith, 1997)
```

```
      DATA TthSK,TthC,CDIL,CSTR,CSW,REGSWL,BLFLL,ALPHA
     >       /33.7,36.8,45.,.1,170.,.222,90.,.1/
C    calculate mean skin temperature from 4 skin sections (nude/clothed; yes/no radiation)
      TSKM=0.
      WSK(1)=(1.-pNUDE(K))*(1.-pRAD(K))
      WSK(2)=(1.-pNUDE(K))*pRAD(K)
      WSK(3)=pNUDE(K)*(1.-pRAD(K))
      WSK(4)=pNUDE(K)*pRAD(K)
      DO 10 I=1,4
          TSKM=TSKM+WSK(I)*TSKA(I)
10 CONTINUE
      IF(TSKM.GT.TthSK)THEN
          WARMS=TSKM-TthSK
          COLDS=0.
      ELSE
          COLDS=TthSK-TSKM
          WARMS=0.
      ENDIF
C    addition of changes in setpoint due to training and acclimation;
C    These are seperated for sweating and blood flow due to the different
C    effects of training and accli. on these parameters:
C    first for blood flow: change of WARMC in WARMCF
      IF(TC.GT.(TthC+OFFFL))THEN
C        drive from warm core for blood flow
          WARMCF=TC-(TthC+OFFFL)
      ELSE
          WARMCF=0.
      ENDIF
C    now for sweating: WARMC becomes WARMCS; as COLDC is not used
C    in bloodflow, it is calculated with the sweating offset changes.
      IF(TC.GT.(TthC+OFFSW))THEN
          WARMCS=TC-(TthC+OFFSW)
          COLDC=0.
      ELSE
          COLDC=(TthC+OFFSW)-TC
          WARMCS=0.
      ENDIF
C    removed mean body drive and replace by core and skin for more clarity:
C    TBM=ALPHA*TSKM+(1.-ALPHA)*TC
C    IF(TBM.GT.TthBM)THEN
C        WARMB=TBM-TthBM
C        COLDB=0.
C    ELSE
C        COLDB=TthBM-TBM
C        WARMB=0.
C    ENDIF
C    changes in calculations for training and acclimation effects
C    use of WARMCF and WARMCS instead of WARMC
      DILAT=CDIL*WARMCF
      STRIC=CSTR*COLDS
C    old:   SKBF=(6.3+DILAT)/(1.+STRIC)
C    a "local" warm skin effect was added (Stolwijk)
      SKBF=XADU*GAINFL*(6.3+DILAT)/(1.+STRIC)*2.0**(WARMS/6.)
      IF (SKBF.LT..5) SKBF=.5
C        Maximal flow=limit*limit change*surface factor
      FLMAX=BLFLL*XFLMAX*XADU
      IF (SKBF.GT.(FLMAX)) SKBF=FLMAX
C    old: alpha in neutral-warm varied between 0.75 and 0.95:
C    ALPHA=.042+.745/(SKBF+.59)
C    new alpha based on body resistance instaed of blood flow only, thus
C    including effect of fat and of muscle blood flow.
C
```

```
      ALPHA=0.08 + 2.*BR
      IF (ALPHA.LT.0.1) ALPHA=0.1
      IF (ALPHA.GT.0.35) ALPHA=0.35
C  remove mean body drive for more clarity; include offset shifts for
C  acclimation and training, as well as change in maximum sweat rate
C  REGSW=CSW*WARMB*EXP(WARMS/10.7)/3600.
C  adjust sweat rate for gain change due to training and acclimation and to Adu
C  REGSW=XADU*GAINSW*CSW*((WARMCS-COLDC)*(1.-ALPHA)+
C  >   ALPHA*(WARMS-COLDS))*EXP(WARMS/10.7)/3600.
C  INSTEAD OF SENSITIVITY DEPENDENT ON ALPHA,
C  INTRODUCE A FIXED SENSITIVITY (CORE=12*SKIN)
      REGSW=XADU*GAINSW*(0.92*CSW*(WARMCS-COLDC)+
     >     .08*CSW*(WARMS-COLDS))*EXP(WARMS/10.7)/3600.
      IF(REGSW.LE..001)REGSW=.001
C  adjust sweat rate MAXIMUM for gain change due to training and acclimation and to Adu
      SWMAX=XADU*REGSWL*XSWMAX
      IF(REGSW.GT.SWMAX) REGSW=SWMAX
C  adjust shivering to body size (lean body mass, relative to
C  standard man:
      SHIV=19.4*COLDS*COLDC*XLBM
      IF(SHIV.GT.(225.*XLBM)) THEN
          SHIV=225.*XLBM
      ENDIF
C  metabolic rate, includes shivering:
      RM=ACT(K)+SHIV
C  respiratory heat loss
      RESP=(.0014*(34.-TA(K))+.0025*(42.-CA))*RM
C  Change heat capacity of tissue according to fat content: old:
C  HCAPSK=ALPHA*W*3500./ADU
C  HCAPC=(1.-ALPHA)*W*3500./ADU
      HCAPSK=ALPHA*W*HSPEC/ADU
      HCAPC=(1.-ALPHA)*W*HSPEC/ADU
C  body core - to -skin resistance
C  old: BR=1./(5.28+.6*1.163*SKBF)
C  Raman factor variable (see Lotens, thesis; later modified by Havenith):
      RAMAN=.25
      IF (TSKM.GT.15.) RAMAN=(TSKM-15.)*.05+.25
      IF (TSKM.GT.25.) RAMAN=(TSKM-25.)*.01+.75
      IF (RAMAN.GT..85) RAMAN=.85
C  New representation of core-to-skin resistance:
C  muscle resistance reduced by muscle blood flow as function of metabolic rate
C  reduction up to factor 5 a 10
      RMUSCLE=0.05/(1+(RM-65.)/130.)
C  fat layer resistance related to layer thickness, including skin (Veicsteinas et al)
      RFATSKIN=0.018*FATTH
C  res. blood flow as in old model
      RSKBF=1/(1.167*RAMAN*SKBF)
C      ADD RESISTANCES
C       MUSCLE + FAT
      RMUFASK=RFATSKIN+RMUSCLE
C      CORE TO SKIN
      BR=1/(1/RSKBF+1/RMUFASK)
C  calculate body heat content/gram body weight
      BHC=((TC-37.)*HCAPC+(TSKM-34.)*HCAPSK)*ADU/W*.001
      RETURN
      END


-Initialisation in main program:

      TRAIN=50.
      ACCLI=0.
      PFAT=15.
```

```
      AGE=25.
      SEX='M'
      FATTH=7.786
      OFFSW=0.
      OFFFL=0.
      GAINFL=1.
      GAINSW=1.
      XFLMAX=1.
      XSWMAX=1.
      VO2 max=40.
      W=75.
      HEIGHT=1.76
      HSPEC=3500.
      LBM=(100.-PFAT)*W/100.
C  size factors
      XLBM=1.          .
      XADU=1.
      XW=1.
      ADU=1.96
      CRIT=.1
      SWCAPSK=2.5
```

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14. SUPPLEMENTARY NOTES

15. ABSTRACT (MAXIMUM 200 WORDS (1044 BYTES))

One of the major gaps in the prediction of heat stress response is the limited implementation of individual characteristics in prediction models. Without this individualization, the evaluation of the resultant average group response prediction necessitates the use of very conservative limit values for body temperature increase. This is caused by the wide range of responses observed within a group.

The present study aimed at the implementation of individual characteristics in a heat stress prediction model (THDYN), in order to investigate whether this would indeed result in a more precise prediction with less variance between predicted and observed responses. For this purpose, the relevant parameters related to anthropometric characteristics (body surface area [A_D], body tissue conductance, body heat capacity), sweating and skin blood flow control (training and acclimation) were introduced in the model. The parameters were derived from literature.

Next, data sets which were not used for the parameter estimation were used for a validation of the model changes. It was found that the individualized model indeed provided an improved prediction. The size of the improvement varied with the climate and the work type however. The best predictions for body heat storage were observed for fixed work loads in a warm humid and in a hot dry climate and for work loads relative to the individual maximum in a warm humid climate (Explained variance 27-53%). For relative work loads in a cool and in a hot dry climate the models predictive capacity for individuals was not significantly improved (<10%).

When this predictive power was compared to that of a multiple regression model derived from the data sets (the "maximal achievable" predictive power), using individual characteristics as independent parameters and body temperature as dependant variable, showed that the independent computer model had between 54 and 89% of the predictive power of the empirical model, except for the cool climate where this was zero.

It can be concluded that the additions to the model provide a good tool for study of effects of individual characteristics, but also that still a substantial part of individual variation is still not understood.

In order to demonstrate the model's possibilities and reactions, several example simulations are presented.

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